AMENDMENT OF THE SPECIFICATION

Please replace the second full paragraph on page 6 with the following amended paragraph.

FIG. 4 shows that caspase-9 is activated by treatment of prostate cancer cells in accordance with an embodiment of the present invention, wherein LNCaP (A) and LNCaP C4-2 (B) cells, respectively, were treated as indicated and caspase-9 activity was determined using ~~calorimetric~~ colorimetric assays. Values are expressed as mean (± SE) caspase activity in units/μg protein (n=4).

Please replace the fourth full paragraph on page 7 with the following amended paragraph.

In one aspect, the present invention comprises a method for inducing cell death in cancer cells, the method comprising treating cancer cells with an effective amount of TRAIL sufficient to induce apoptosis in at least a portion of the treated cancer cells. As used herein, TRAIL may comprise a wild-type trail polypeptide having the amino acid sequence of SEQ ID NO: 1 (Pitti et al., 1996), or a biological equivalent thereof.

Please replace the third full paragraph on page 18 with the following amended paragraph.

The present invention relies on the discovery that Mifepristone can increase the efficacy of TRAIL in inducing apoptosis in those prostate cancer cells which are resistant to the apoptic effects of TRAIL. Thus, as shown in FIG. 1 (Panels B and D), treatment of LNCaP C4-2 prostate cancer cells with 400 ng/ml TRAIL reduces cell survival as early as 8 hour and continues with exposure to the drug. Thus, LNCaP C4-2 cells are relatively sensitive to the effects of TRAIL. In contrast, treatment of LNCaP cells with 400 ng/ml TRAIL does not alter cell survival significantly (FIG. 1, Panels A and C). Treatment of LNCaP cells with Mifepristone followed by TRAIL, however, results in a significant

decrease in cell survival. Treatment of both LNCaP and LNCaP C4-2 cells with Mifepristone alone has little effect on survival (FIG 1).

Please replace the paragraph that begins on the bottom of page 22 and ends on the top of page 23 with the following amended paragraph.

NFκB is an important member of survival pathway (M.W. Mayo and A.S. Baldwin, Biochim. Biophys. Acta, **1470**, M55-M62, 2000). NFκB is involved in transformation and tumorigenesis and also suppresses apoptotic pathways. NFκB is sequestered in the cytoplasm in an inactive state by its interaction with IκB protein. Upon phosphorylation of Iκβ, followed by ubiquitination and degradation, NFκB translocates into the nucleus, where it induces transcriptional activity of target genes. NFκB can block TNFα - or TRAIL- induced apoptosis by influencing the function of DcR1, RIP (receptor interacting protein), FADD and caspase-8 (Wang, C-Y., et al, Science **281**, 1680-1683, 1998; Sugiyama, H. J., et al., Biol. Chem., **274**, 19532-19537, 1999; Hu, W-H., et al., J. Biol. Chem., **275**, 10838-10844, 2000; Jones, D.R., et al., Ann. Thoracic Surg., **70**, 930-937, 2000; K. Kuwano and N. Hara, Am. J. Respir. Cell Mol. Biol., **22**, 147-149, 2000; Lin, Y., et al., Mol. Cell. Biol., **20**, 6638-6645, 2000; Nagaki, M., et al., Hepatol., **32**, 1272-1279, 2000; Bernard, D., et al., J. Biol. Chem., **276**, 27322-27328, 2001). Furthermore, NFκB may indirectly ~~affected~~ affect apoptosis through Inhibitor of Apoptosis Proteins, cIAP1, cIAP2 and XIAP (Wang, C-Y., et al, Science **281**, 1680-1683, 1998; Chu, Z-L., et al., Proc. Natl. Acad. Sci., USA, **94**, 10057-10062, 1997; Van Atwerp, D.J., et al., Trends Cell Biol., **8**, 107-111, 1998; Erl, W., et al., Circ. Res., **84**, 668-677, 1999; M. Holcik and R.G. Korneluk, Nature Rev. Mol. Cell Biol, **2**, 550-556, 2001; Levkau, G., et al., Circ. Res., **88**, 282-290, 2001), which inhibit initiator and effector caspases (M. Holcik and R.G. Korneluk, Nature Rev., Mol. Cell Biol., **2**, 550-556, 2001; Suzuki, Y., et al., J. Biol. Chem., **276**, 27058-27063, 2001). NFκB also blocks apoptosis by increasing the expression of Bcl_{XL}, an antiapoptotic protein (Ravi, R., Nature Cell Biol., 3, 409-416, 2001).

Please replace the paragraph that begins on the bottom of page 24 and ends on the top of page 25 with the following amended paragraph.

In addition, genetic constructs, such as transcription factor decoys (TFDs) or adenoviral construct comprising mutated IkBM as described herein ~~(Example9)~~ Example 9 may be delivered into the cell by infection (e.g. as with ~~recombinate~~ recombinant adenovirus), direct transfection of naked (unprotected) DNA or may employ a type of carrier, such as liposomes. For example, *in vivo* delivery of TFDs using a hemaglutinating virus of Japan (HVJ)-liposome carrier (Morishita *et al.*, *Nature Med., 3*: 894-899, 1997) or a Sendai virus-liposome carrier (US Patent No. 6,262,033) has been described in a mycocardial infarct model. Using HVJ liposomes, infusion of fluorescently labeled NF-κB TFDs into the left coronary artery resulted in fluorescence in coronary microvascular endothelial cells with a reduction of infarct size and reduced levels of IL-6 and VCAM mRNA. Recombinant adenoviruses have been shown to achieve high ~~effecienty~~ efficiency gene transfer after direct, in vivo delivery to airway epithelium pepatocytes, vascular endothelium, CNAS parenchyma and a number of other tissues (e.g. La Salle, Science **259**, 988-990, 1993; Gomez-Foix, J. Biol. Chem., **267**, 25129-25134, 1992; Rich, Human Gene Therapy, **4**, 461-476, 1993; Guzman, Circulation Res., **73**, 1201-1207, 1993; Bout, Human Gene Therapy, **5**, 3-10, 1994).